ORIGINAL ARTICLE

Electromyogram–Lengthening Velocity Relation in Plantar Flexors During Stance Phase of Gait in Patients With Hypertonia After Acquired Brain Injury

John W. Chow, PhD, Stuart A. Yablon, MD, Dobrivoje S. Stokic, MD, DSc

ABSTRACT. Chow JW, Yablon SA, Stokic DS. Electromyogram–lengthening velocity relation in plantar flexors during stance phase of gait in patients with hypertonia after acquired brain injury. Arch Phys Med Rehabil 2012;93:2287-94.

Objective: To examine the velocity-dependent change in medial gastrocnemius (MG) activity during the stance phase of gait in patients with moderate to severe resting hypertonia after stroke or traumatic brain injury (TBI).

Design: Cohort study.

Setting: Motion analysis laboratory in a tertiary-care rehabilitation hospital.

Participants: Convenience sample of patients with chronic TBI and stroke (n=11 each), and age- and sex-matched healthy controls (n=22).

Intervention: Not applicable.

Main Outcome Measures: Frequency and gain (steepness) of positive (>0) and significant positive (>0 and goodness of fit $P \leq .05$) electromyogram–lengthening velocity (EMG-LV) linear regression slope in MG during the stance phase of gait.

Results: Positive and significant positive slopes were found significantly more often on the more affected (MA) than less affected (LA) side in patients with TBI but not stroke. Both the frequencies of positive and significant positive slopes on the MA side in patients with TBI were also significantly higher than in controls. However, neither the gain of positive nor significant positive EMG-LV slope was different between the MA and LA sides or in comparison with controls. Positive slope parameters were not related to Ashworth score on the MA side.

Conclusions: The frequency and gain of positive EMG-lengthening slope did not effectively differentiate patients from controls, nor were they related to the resting muscle hypertonia. Motor output during MG lengthening in the stance phase of gait is apparently not exaggerated or related to resting hypertonia in patients with chronic TBI and stroke. Thus, changes in gait during stance cannot be ascribed to increased stretch reflex activity in MG muscle after acquired brain injury.

Key Words: Reflex, stretch; Rehabilitation; Stroke; Traumatic brain injuries.

SPASTICITY IS COMMONLY defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex as one component of the upper motor neuron syndrome."[1(p485)] In addition to hyperactive stretch reflexes, other positive features of the upper motor neuron syndrome include muscle spasms, abnormal muscle coactivation, clonus, and spastic dystonia.[2,3] Spasticity is ascribed to an unopposed increase in excitatory over inhibitory input to the α-motor neurons[3-5] leading to muscle overactivity.[6] Although spasticity is associated with altered control of many spinal reflex pathways,[7] implications for purposeful, active motor function are still debated.[7,8]

Gait impairments are common in patients with muscle hypertonia in the lower extremities after stroke or traumatic brain injury (TBI).[9-13] Opposing views, however, exist relative to the contribution of spasticity in its strict sense to gait dysfunction. Early electromyogram (EMG) studies during treadmill walking only revealed greater activity in the tibialis anterior (TA) muscle during swing in patients with spinal spasticity compared with healthy controls.[14] This suggested changes in muscle mechanical properties as a possible culprit.[15,16] Ada et al[8] simulated a single support phase of gait in the sitting position by rotating the foot at different frequencies with the knee extended while the subject was asked to activate the gastrocnemius muscle at 10% of the maximum EMG level. They reported no difference in stretch-induced EMG between 14 stroke patients (5–20mo postonset) and healthy controls, and concluded that stretch reflex activity during walking is not increased after stroke. However, interventions that reduce stretch reflex excitability, such as intrathecal administration of baclofen, may improve,[17-19] worsen,[20] or variably affect many gait parameters.[21,22] Thus, further studies to delineate features of spasticity during gait are warranted to guide selection of patients, interventions, and outcome measures.

List of Abbreviations

ABI	acquired brain injury
AS	Ashworth score
CMRR	common-mode rejection ratio
EMG	electromyogram
EMG-LV	EMG-lengthening velocity
LA	less affected
L_S	shank length
MA	more affected
MG	medial gastrocnemius
TA	tibialis anterior
TBI	traumatic brain injury

From the Center for Neuroscience and Neurological Recovery, Methodist Rehabilitation Center, Jackson, MS (Chow, Stokic); and Brain Injury Program, Glenrose Rehabilitation Hospital, Edmonton, Alberta, Canada (Yablon).

Presented in part to the Society for Neuroscience, October 17–21, 2009, Chicago, IL.

Supported in part by the Wilson Research Foundation, Jackson, MS.

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit on the authors or on any organization with which the authors are associated.

Reprint requests to John W. Chow, PhD, Methodist Rehabilitation Center, 1350 East Woodrow Wilson, Jackson, MS 39216, e-mail: *jchow@mmrcrehab.org*.

In-press corrected proof published online on May 15, 2012, at www.archives-pmr.org.

0003-9993/12/9312-01233$36.00/0
http://dx.doi.org/10.1016/j.apmr.2012.03.020

Crenna[23(p572)] introduced the concept of "dynamic assessment of spasticity during walking" on the premise that "the higher the effectiveness of velocity-sensitive excitatory reflex components, the tighter would be the correlation between motor output and velocity of muscle lengthening."[23(p572)]

This was determined by plotting EMG against muscle lengthening velocity during walking in patients with cerebral palsy. Based on this concept, Lamontagne et al[24(p1696)] developed a "locomotor-specific measure of spasticity" in ankle plantar flexors during the stance phase of gait, with EMG-lengthening velocity (EMG-LV) slope as an outcome measure (called "spasticity index" in Lamontagne[25] and Lin[26] and colleagues).

When averaged over all gait cycles, the gain of EMG-LV slope in the plantar flexors during stance was found positive (>0) on the paretic side in 20 of 30 subjects within 6 months of stroke, but not on the nonparetic side or in controls. The average gain of EMG-LV slope correlated positively with the Ashworth score (AS) in the paretic plantar flexors and negatively with gait speed.[25] Lin[26] found that EMG-LV slope in the paretic leg was positive in 44% of gait cycles from 68 stroke subjects (7d to 31y poststroke). The spasticity index, calculated as the average gain of positive slopes only, was found significantly correlated to most temporospatial parameters of gait. Both Lamontagne[24,25] and Lin[26] interpreted positive EMG-LV slope as the evidence of hyperactive stretch reflexes during lengthening of plantar flexors in the stance phase of gait.

The purpose of this study was to further characterize the velocity-dependent increase in medial gastrocnemius (MG) activity in the stance phase of gait in patients with acquired brain injury (ABI) with moderate to severe lower limb muscle hypertonia. We replicated the previously used approach[24-26] for comparison purposes, but put special emphasis on the strength of the linear association between EMG and lengthening velocity, in accordance with the concept of Crenna.[23] Thus, our specific aim was to compare the frequency and gain of positive EMG-LV slope in MG muscle during stance between patients with ABI and age- and sex-matched healthy controls. We hypothesized that the EMG-LV slope would be more often positive and steeper on the more affected (MA) side of patients compared with their less affected (LA) side and healthy controls. The associations with gait speed and resting muscle hypertonia were explored in secondary analyses.

METHODS

Participants

A convenience sample of 11 stroke and 11 TBI patients was recruited from a spasticity and motor disorders clinic (table 1). The inclusion criteria were a significant increase in lower limb muscle hypertonia that impairs function or care, and the ability to walk safely faster than 10cm/s for at least 10m with or without assistive devices. Wearing a short, nonrigid polypropylene ankle-foot orthosis to prevent foot drop during gait was permitted because it allowed passive dorsiflexion during the stance phase, as verified by ankle range of motion. Patients with evidence of ankle clonus during the stance phase were excluded to eliminate the confounding effect of the rhythmic EMG pattern to MG activity. Because of the age difference between stroke and TBI patients, 2 age- and sex-matched healthy control groups were recruited (n=11 each). Control subjects in the stroke-matched group (mean ± SD: age, 41±9y; height, 170±14cm; body mass, 76±22kg) and TBI-matched group (mean ± SD: age, 29±10y; height, 174±14cm; body mass, 76±22kg) reported no orthopedic and neurologic disorders at the time of testing. Each subject attended 1 data collection session and signed the informed consent approved by our institutional review board for human research.

Experimental Setup and Instrumentation

Gait data were collected at 60Hz with 8 digital Hawk cameras[a] surrounding a 7-m-long walkway. Five forceplates[b] concealed and flush in the middle of the walkway were used to determine critical instants of gait (sample rate, 1200Hz). Passive spherical reflective markers were affixed to body landmarks according to the Helen Hayes marker system.[27]

Bipolar surface EMG electrodes with built-in preamplification[c] (model MA411; gain 20, 2-cm center-to-center distance, input impedance $>10^{10}\Omega$, common-mode rejection ratio [CMRR] >100dB) were attached to MG and TA muscles bilaterally, using electrode placement guidelines described in Cram and Kasman.[28] EMG data of TA were used in a companion study.[29] Signals were further amplified by an EMG system[c] (model MA300; input impedance, 31KΩ, CMRR >50dB) before 12-bit analog-to-digital conversion (sampling rate, 1200Hz). Video, EMG, and ground reaction force (when available) data were collected synchronously on the EVaRT data acquisition system.[a]

Experimental Protocol

The subject walked 8 to 10 times along the walkway with short pauses in between with customary shoes and assistive devices, if any. Patients were instructed to walk at a self-selected free speed, and controls at a self-selected very slow speed, which provides more appropriate comparison between patients with ABI and controls[9] and differentiates EMG-LV features that are disorder-specific from walking at a slow speed. Very slow rather than patient-matched speed was chosen for controls to ensure natural walking. No instruction was given about stepping on the forceplates. Data acquisition started after the subject took a few steps and terminated before the end of the walkway. Thus, each trial included a steady-state gait.

Before recording gait in patients, muscle hypertonia was assessed by a physical therapist in the bilateral hip flexors and extensors, knee flexors and extensors, and ankle plantar flexors, using the Modified Ashworth Scale[30] (see table 1). Despite its limitations,[31] the Modified Ashworth Scale is customarily used

Table 1: Characteristics of TBI and Stroke Subjects

Injury Type	Age (y)	Height (cm)	Mass (kg)	Men/Women	Time Postonset (mo)	Ankle-Foot Orthosis	Cane	Average AS‡		Plantar Flexor AS	
								MA Side	LA Side	MA Side	LA Side
TBI	27±11	175±11	79±24	7/4	38±29	4	5	2.1±0.5†	1.1±0.3	3.2±0.8*	1.4±0.7
Stroke	41±9	169±13	95±15	3/8	45±46	8	6	2.0±0.2†	1.0±0.0	3.4±0.5†	1.0±0.0

NOTE. Values are mean ± SD or n.
*$P<.05$, †$P<.01$; significant difference between MA and LA sides.
‡Average for hip flexors and extensors, knee flexors and extensors, and ankle plantar flexors.